SCIENTIFIC LETTER

Triggering of acute coronary syndromes after a chemical plant explosion

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The impact of an industrial disaster has scarcely been studied in cardiology. On 21 September 2001 at 10.17 am, a chemical plant storing ammonium nitrate exploded in the urban area of Toulouse, France. The explosion induced an earthquake reaching 3.4 on the Richter scale; 30 people died, 3000 were injured, and 27 000 homes were destroyed. The explosion released a cloud of atmospheric pollutants essentially composed of nitrogenous compounds. During a period of 24 hours, the greatest concentrations of NO_2 measured by the two ambient stations of an air quality network located near the chemical plant varied from 20–95 μg/m^3. Another urban station recorded once, at 10.30 am, a concentration of 388 μg/m^3. The monitor measuring the release of specific chemicals (NH_3) was destroyed by the explosion. No other data were available except for the personal detector devices of the firemen working around the crater (a perimeter of 500 m).

Since 1985 the acute myocardial infarction (AMI) incidence has been systematically recorded in the population living in the explosion area.[1] We investigated the relation between this stressful event and the occurrence of acute coronary syndromes (ACS). The main objective was to test whether this industrial disaster has led to an increased incidence of ACS in the days after the explosion.

METHODS

The target area was defined as administrative districts located within a radius of 3 km of the epicentre of the explosion. The study population, aged 35–74 years and living in the target area, comprised 181 944 inhabitants in 2001 and 179 075 in 2000.

Three definitions of ACS were used: definite AMI plus coronary death (definition 1), definite AMI plus coronary death plus sudden death (definition 2), and definite AMI plus coronary death plus sudden death plus death with insufficient data (definition 3).[1]

Briefly, for definite AMI, clinical, ECG, and enzymatic data were available. Coronary deaths, sudden deaths, or deaths with insufficient data were validated by all available medical and forensic reports, as well as interviews with general practitioners. Coronary death corresponded to rapid death after chest pain (typical or atypical symptoms) or a known history of coronary heart disease with no clear evidence supporting any other cause of death. Sudden death corresponded to rapid death (less than one hour) without symptoms, a personal history of coronary heart disease, or evidence supporting another cause of death. Death with insufficient data was regarded as a death with no symptoms (or inadequately described), no history of coronary heart disease, and no other diagnosis.[1]

The number of ACS occurring after the explosion was compared with ACS incidence during a reference period of similar length. The reference period was defined as the average of three periods, each with a length equivalent to that of the target period: one just before the explosion on 21 September 2001, one just before 21 September 2000, and one just after 21 September 2000.

The relative risk was estimated with the standardised incidence ratio (SIR)—that is, the ratio of the number of ACS observed (D) to the number expected (E).

We used the following normal approximation to the Poisson distribution (Z statistic):

$$Z = \sqrt{9\tilde{D}}\left\{1 - \frac{1}{9\tilde{D}} - \left(\frac{E}{\tilde{D}}\right)^{1/3}\right\}$$

where $\tilde{D} = D$ if D exceeds E and $\tilde{D} = D + 1$ otherwise.[2]

Three target periods of different lengths were investigated to evaluate the time lag related to the occurrence of events: a three day period, a five day period, and a 10 day period.

RESULTS

The number of ACS was significantly higher after the explosion than during the reference period, whatever the definition of ACS used. The SIR decreased from the three day to the 10 day period (table 1).

DISCUSSION

Statistical analysis of data around 21 September 2001 shows evidence for a probable excess of ACS during the first few days after the explosion, about 3.5 higher than the usual incidence of ACS during an equivalent period. ACS occurred in the whole exposed population, whatever the sex, age, or personal medical history of the inhabitants. None of the rescue workers had a cardiac event during the monitoring period.

The results are consistent with several published reports about the short term effects of a stressful event on cardiovascular morbidity and mortality. The relation between a transient stressful event and an increased occurrence of ACS is relatively constant, regardless of the population and the nature of the triggering event: war,[3] earthquake,[4] or sporting event.[5]

Other factors are highly unlikely to have induced an increase of ACS after the explosion. No other contemporary environmental event occurred during this period. Meteorological variables varied within the usual range observed (from 5.6–16.8℃ for the minimum temperature and from 18.6–26.3℃ for the maximum). Exposure concentrations of pollutants measured during a few hours after the explosion were likely to provoke only transient irritant effects of mucous membranes. Moreover, SIRs calculated for the population living outside the target area were not significant; for the three days after the explosion, SIRs were as follows for definitions of ACS 1, 2, and 3: 0.7 (95% confidence interval

Abbreviations: ACS, acute coronary syndromes; AMI, acute myocardial infarction; CI, confidence interval; SIR, standardised incidence ratio

Table 1 Number of acute coronary syndromes and standardised incidence ratio during the target period (21 September 2001) and an equivalent period of equal length in 2000

	Duration of reference and target periods								
	3 days			5 days			10 days		
	Def 1	Def 2	Def 3	Def 1	Def 2	Def 3	Def 1	Def 2	Def 3
Number of events									
Reference period (total of 3 periods)	4	5	5	9	10	11	19	21	26
Before 21 Sept 2000	3	3	3	5	5	6	8	9	11
After 21 Sept 2000	0	0	0	3	3	3	5	5	6
Before 21 Sept 2001	1	2	2	1	2	2	6	7	9
Target period									
After 21 Sept 2001	5	6	6	10	11	13	11	14	17
Standardised incidence ratio	3.8	3.6	3.6	3.3	3.3	3.6	1.7	2.0	2.0
95% Confidence interval	1.2 to 8.8	1.3 to 7.8	1.3 to 7.8	1.6 to 6.1	1.7 to 5.9	1.9 to 6.1	0.9 to 3.1	1.1 to 3.4	1.1 to 3.1
Deaths at 28 days									
Reference period	0	1 (20%)	1 (20%)	0	1 (10%)	2 (18%)	1 (5%)	3 (14%)	8 (31%)
Target period	1 (20%)	2 (33%)	2 (33%)	1 (10%)	2 (18%)	4 (31%)	2 (18%)	5 (35%)	8 (47%)

Definition (Def) 1: definite acute myocardial infarction (AMI) + coronary death; definition 2: definite AMI + coronary death + sudden death; definition 3: definite AMI + coronary death + sudden death + death with insufficient data.
Reference period: number of events for the three periods, all of equivalent length to the target period: one just before the explosion on 21 September 2001, one just before 21 September 2000, and one just after 21 September 2000.

(CI) 0.2 to 1.9), 0.6 (95% CI 0.1 to 1.8), and 0.6 (95% CI 0.1 to 1.6), respectively. Lastly, seasonal variability may originate from the excess of ACS observed on 21 September and afterwards. SIRs for the first years of the registry (1985–1993) and for a 10 day time lag after 21 September vary from 0.9 (95% CI 0.5 to 1.6) for the minimum to 1.2 (95% CI 0.8 to 1.9) for the maximum.

The sensitivity analysis based on a range of ACS definitions from the most restrictive (definition 1) to the least restrictive (definition 3) showed a consistent association between the explosion and ACS occurrence.

Increased occurrence of ACS after a major industrial disaster can be attributed to acute stress caused by the explosion, as well as to a subchronic emotional impact during the next days due to the destruction of homes and the material difficulties that are encountered.

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